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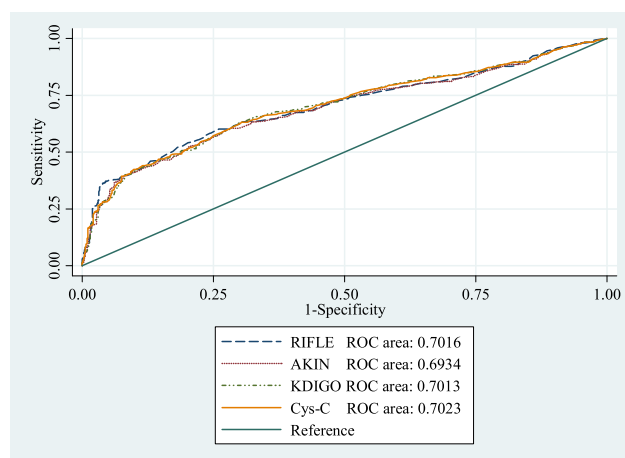
**Comparison of Novel Definitions (RIFLE, AKIN, KDIGO and Cys-C criteria) for Acute Kidney Injury Among Critically Ill Patients**J. J. Zhou<sup>1</sup>, Y. Liu<sup>2</sup>, P. Fu<sup>1</sup><sup>1</sup>West China Hospital of Sichuan University, Chengdu, China<sup>2</sup>Chengdu Integrated TCM & Western Medicine Hospital, Chengdu First People's Hospital, Chengdu, China

**Objective:** AKI is a major clinical problem and predictor of prognosis in critically ill patients, whatever any new definitions was used. Our study was to determine if the new Cys-C (Cystatin C) criteria for identification and short-term prognosis of AKI was superior to the previous RIFLE (Risk, Injury, Failure, Loss of kidney function, and End-stage renal failure), AKIN (Acute Kidney Injury Network), and Kidney Disease: Improving Global Outcomes (KDIGO) criteria.

**Methods:** A cohort of 4642 patients admitted to five intensive care units (ICUs) was analyzed in this retrospective and multicenter study. AKI occurring during whole ICU stay was identified by the RIFLE, AKIN, KDIGO and Cys-C criteria, and discriminative ability of each AKI stage for the prediction of 28-day mortality was assessed. Receiver operating curve (ROC) were applied to compare the predictive ability for mortality, and logistic regression analysis was used for the calculation of odds ratios (OR) and 95% confidence intervals (CI).

**Results:** In the 1036 patients enrolled, the incidences of AKI were 26.4%, 34.1%, 37.8% and 36.1%, respectively, under the RIFLE, AKIN, KDIGO and Cys-C criteria. Patients with AKI had higher mortality and longer length of stay in ICU than those without in all definitions. Concordance in AKI diagnosis between Cys-C and KDIGO criteria was 95.9%, higher than AKIN and RIFLE criteria ( $p < 0.0001$ ). The areas under the ROC curves were 0.7016 for RIFLE, 0.6934 for AKIN, 0.7013 for KDIGO, and 0.7023 for the Cys-C criteria ( $p < 0.05$  for all). Cys-C criteria were significantly greater discrimination than the other three criteria ( $p < 0.05$ ).

**Conclusion:** KDIGO criteria identified significantly more AKI than RIFLE or AKIN or Cys-C criteria. The mortality of AKI had significantly higher than no-AKI patients according to all four criteria. The new Cys-C criteria were more excellent than the other three criteria in predicting short-term outcomes in critically ill patients.



**Figure 1.** Receiver operating characteristic (ROC) curve and the discriminatory power for mortality in critically ill patients for the RIFLE, AKIN, KDIGO, and Cys-C criteria.

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**Significance of Blood Perfusion in Treatment of Acute Intoxication Induced by Drug and Poison**

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**Objective:** To investigate the effect of blood perfusion (HP) in the treatment of acute poisoning and its clinical effect.

**Methods:** The clinical data of 86 patients with oral poisoning were retrospectively analyzed. The patients were divided into HP treatment group (45 cases) and conventional treatment group without HP treatment (41 cases). All patients were given gastric lavage treatment. The HP treatment group patients were given routine coagulation tests. According to these results the first time administration dosage of heparin was determined as 0.5–1.0 mg/kg, and additional dosage was 10–20 mg/h. At the same time pay attention to monitoring the blood coagulation and platelets, blood flow velocity was set at 150–180 ml/min, and activated carbon canister was replaced every 2 hours, and repeated blood perfusion treatment on severe poisoning patients. Two groups of patients were both given conventional drug treatment.

**Results:** Compared to the control group, the cholinesterase activity recovery time and length of hospital stay in HP group were significantly shorter ( $P < 0.05$ ), but because of the application of heparin, 8 patients in HP group had transient coagulation disorders, which recovered without special treatment.

**Conclusion:** HP in the treatment of all kinds of poisoning curative effect can be significantly reduced the number of days hospitalized patients and reduce the mortality of patients, some poisoning patients with no specific antidote, in treatment shows unique advantages, can significantly improve the success rate of rescue.

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**Liver X Receptor Agonist TO901317 Protects Mice Against Cisplatin-induced Kidney Injury**

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**Objective:** Liver X receptors (LXRs) are in the nuclear receptor superfamily and are contained in the regulation of lipid and cholesterol metabolism. Besides, LXRs are considered crucial regulators of the inflammatory response and innate immunity. The current study evaluates the *in vivo* effects that the synthetic LXR agonist TO901317 protects against cisplatin-induced kidney injury in mice.

**Methods:** Mice received cisplatin administration through a single intraperitoneal (ip) injection (20 mg/kg in saline). And then the mice were treated with TO901317 by daily gavage (10 mg/kg/d) 12 hours post cisplatin administration and cisplatin nephrotoxicity was evaluated.

**Results:** At 72 hours after cisplatin treatment, elevated plasma urea and creatinine levels ( $P < 0.05$ ) were evidenced which indicates the renal dysfunction of the vehicle treated mice, consistent with tubular necrosis, protein cast, dilation of renal tubules, and desquamation of epithelial cells in renal tubules. In contrast, the severity of renal dysfunction and histological damage was reduced in TO901317 treated mice ( $P < 0.05$ ). In accordance, circulating tumor necrosis factor alpha (TNF- $\alpha$ ) levels, renal TNF- $\alpha$ , p47<sup>phox</sup>, gp91<sup>phox</sup>, and protein expression levels and COX-2 mRNA, renal MCP-1, VACAM-1 mRNA and ICAM-1 contents, and renal PGE<sub>2</sub> amounts, were higher in samples from cisplatin treated mice in comparison with controls ( $P < 0.05$ ), but attenuated in the TO901317 treatment group ( $P < 0.05$ ).

**Conclusion:** Taken together, treatment with the LXR agonist TO901317 ameliorated the inflammatory response and oxidative stress in cisplatin-induced kidney injury in mice.

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**Inhibition of PKC $\delta$  Protects Against Cisplatin-induced Renal Tubular Cell Apoptosis by Activation of Autophagy Through Direct Inhibition of AKT/ mTOR Signalling**Dongshan Zhang<sup>1,2</sup>, Zheng Dong<sup>1,2</sup><sup>1</sup>Department of Nephrology, Second Xiangya Hospital, Central South University, Changsha, Hunan, China<sup>2</sup>Department of Cellular Biology and Anatomy, Medical College of Georgia, Georgia Regents University and Charlie Norwood VA Medical Center, Augusta, Georgia, USA